before the insertion of a penile prosthesis (diabetes, nephropathy).

With the advent of these approaches, it has become clear that physical disability does not necessarily lead to sexual disability. JULIE G. BOTVIN MADORSKY, MD

REFERENCES

Bray GP, DeFrank RS, Wolfe TL: Sexual functioning in stroke survivors. Arch Phys Med Rehabil 1981 Jun; 62:286-288
Comfort A: Sexual Consequences of Disability. Philadelphia, GF vivors. Arch Phys Med Rehabil 1981 Jun; 62:286-288
Comfort A: Sexual Consequences of Disability. Philadelphia, GF
Stickley, 1978
Madorsky JGB: Sexuality of the physically disabled. Med Asp Hum
Sex 1983 Jul; 17:45-53
Madorsky JGB, Dixon TP: Rehabilitation aspects of human sexuality.
West J Med 1983 Aug; 139:174-176
Ward-McKinlay T, Madorsky JGB, Ward-McKinlay C: Sexuality and
disability, chap 7, In Hansen JC, Woody JD, Woody RH (Eds): Family
Therapy Collections—Sexual Issues. Rockville, MD, Aspen Systems, 1983,
pp. 129-152

Therapy C pp 129-152

## **Urinary Infection in a Neurogenic Bladder**

NEUROGENIC BLADDER DYSFUNCTION can occur due to traumatic and other lesions of the spinal cord such as multiple sclerosis, tumors, diabetes mellitus, stroke and the like. The resulting bladder dysfunction causes disturbances in normal micturition. In many patients with such dysfunction, use of an indwelling catheter may be eliminated by intermittent catheterization, therapy with α-sympathetic blockers or transurethral surgical repair (or all three). Bacterial colonization or obvious urinary tract infection continues to be a common occurrence, particularly in patients who wear external collecting devices and a leg bag. Urinary tract infections are a major cause of mortality in patients with spinal cord injury and neurogenic bladders.

Diagnosing urinary tract infections by symptoms alone in patients with neurogenic bladders is unreliable. Lack of symptoms such as fever, chills, dysuria and flank pain, in spite of significant bacteriuria (greater than 100,000 per ml), continues to be common in most patients with spinal cord injury who have unobstructed voiding. In an unpublished study by Perkash and coworkers (1982), only 3% of 103 patients with spinal cord injury had symptoms, in spite of bacteria in a urine specimen numbering more than 100,000 per ml. Thus, symptoms per se are poorly correlated with significant bacteriuria in such patients. Due to this lack of clinical signs, bacteriuria poses a therapeutic dilemma in treatment and management. In addition, with complicating factors of perineal and drainage bag contamination, the question of colonization versus true tissue invasion arises. Quantitating urine pyuria has proved helpful for clinically determining urinary tract infection. Microscopic examination of urine sediment for the presence of leukocytes is a time-honored technique, but one that is poorly standardized and thus semiquantitative. Quantitating pyuria, however, is a simple and logical improvement of this technique. The usefulness of hemacytometer counting of uncentrifuged urine leukocytes in urine specimens of a general hospital's inpatients was reported in 1976. The presence of 10,000 or more leukocytes per ml of urine was correlated with the presence of a urinary tract infection. In another (unpublished) study, the presence of more than 10,000 leukocytes per ml of urine was highly predictive of the presence of bacteriuria in men with spinal cord injury.

As a result of our continued investigations to define and manage urinary tract infection in a neurogenic bladder, certain general guidelines have been developed. For patients being maintained on indwelling Foley catheter drainage, only symptomatic bacteriuria associated with fever and chills is treated; otherwise, no antimicrobial treatment or prophylaxis is recommended. Patients catheterized intermittently who remain asymptomatic despite significant bacteriuria are not generally treated. Due to regular, timed bladder emptying, bladder bacteriuria, if due to simple colonization, could be transient and thus flushed out at the next catheterization. However, if a patient has bacteriuria and symptoms of fever and chills, treatment is based on the urine culture-sensitivity report. Finally, patients receiving external condom drainage who have asymptomatic bacteriuria with fewer than 10,000 leukocytes per ml of urine may not require treatment if the bladder is draining adequately. This type of bacteriuria could be a contaminant from a patient's own perineum or drainage bag. Attention towards maintaining proper hygiene might be the only recourse for reducing or controlling the incidence of asymptomatic bacteriuria.

The recent development and use of noninvasive "markers" to define a site of infection and therefore direct the most effective type of treatment is now being studied. Surface blockers or specific antipilial antibodies available to prevent bacterial adhesion and colonization on the bladder mucosa may provide a lasting cure.

INDER PERKASH, MD JAN GIROUX, RN

REFERENCES

Graham SD: Present urological treatment of spinal cord injury patients.

J Urol 1981 Jul; 126:1-4

Källenius G, Svenson S, Möllby R, et al: Structure of carbohydrate part of receptor on human uroepithelial cells for pyelonephritogenic Escherichia coli. Lancet 1981 Sep 19; 2:604-606

Rhame F, Perkash I: Urinary tract infection occurring in recently injured spinal cord injury patients on intermittent catheterization. J Urol, in press

in press Roberts JA: Pathogenesis of pyelonephritis. J Urol 1983 Jun: 129:

## **Electrodiagnosis in Brachial Plexus Injury**

FOLLOWING brachial plexus injury, electrodiagnostic changes are seen most commonly during needle electromyography. Changes in nerve conduction velocity studies are much less common because, one, plexus injuries most often produce axonal loss rather than demyelination and, two, there are many technical problems with brachial plexus nerve conduction studies.

The electromyographic examination must include the infraspinatus and supraspinatus, rhomboid, levator scapulae, serratus anterior and cervical paraspinal muscles to accurately assess the proximal extent of plexus injury.

Although changes in motor or sensory conduction velocities are not common in plexopathy, a reduction in amplitude of the motor or sensory evoked potential is common. Abnormalities of conduction or evokedpotential amplitude change may be most common in the musculocutaneous nerve. The role of F-wave stud-